*British Heart Journal*, 1975, 37, 445-448.

# Changing haemodynamics in patient with papillary muscle dysfunction

W. Markiewicz,[1] S. Amikam, N. Roguin, and E. Riss

*From the Department of Cardiology, Rambam University Hospital, Haifa, and the Abba Khoushy Medical School, Israel*

*A patient with papillary muscle disease caused by myocardial infarction was studied before and after injection of phenylephrine. The pulmonary wedge pressure was normal at rest. However, pressures and murmur changes, occurring spontaneously and after injection of phenylephrine, suggested that intermittent severe mitral regurgitation contributed significantly to the recurrent episodes of acute left heart failure presented by this patient.*

The syndrome of mitral regurgitation caused by papillary muscle dysfunction has been recognized with increasing frequency within the last decade. Though many studies have described spontaneous and drug-induced changes of the systolic murmur, few document acute haemodynamic changes in this syndrome (Brody and Criley, 1970; Lipp *et al.*, 1972). This prompted us to report the following case.

## Case report

A 53-year-old woman was admitted with a 6-month history of recurrent pulmonary oedema associated with chest pain. Except for mild diabetes, she had been previously healthy. Between the episodes of acute left heart failure, she was feeling relatively well, complaining only of atypical chest pain and of mild dyspnoea on effort.

*Clinical examination* on admission showed a woman in no distress. Her blood pressure was 110/70 mmHg (14.6/9.3 kPa) and the pulse was regular, 70/minute. The jugular pulse and pressure were normal, the edge of the liver was palpated 3 cm under the costal margin, and no peripheral oedema was present. The lungs were clear. The left ventricle was enlarged on palpation and a prominent lower left sternal impulse was felt. The first heart sound was reduced in intensity and the pulmonary closure sound was accentuated. A grade 3/6 pansystolic murmur was heard at the apical area, conducted to the axilla, and along the left sternal border, followed by a third heart sound and a short mid-diastolic rumble. The murmur varied only slightly from day to day.

*The electrocardiogram* showed a prominent R wave in lead V1 and a qRS pattern in leads V5-V6 with pronounced right axis deviation. It was thought to be compatible with an old anterolateral and true posterior myocardial infarction, or with pronounced right ventricular hypertrophy. P wave abnormalities suggesting left atrial enlargement were present and the PR interval was 0.20 s.

*The chest* x-*ray* showed moderate enlargement of the left ventricle and left atrium. There were signs of pulmonary venous congestion and of pulmonary arterial hypertension.

Acute subvalvar mitral regurgitation was considered to be the most likely diagnosis at this stage.

*Right heart catheterization* was performed under local anaesthesia. Pethidine 50 mg and promethazine hydrochloride 50 mg were given intramuscularly 1 hour before catheterization. No shunts were detected. The pulmonary artery pressure was 56/24 mmHg (7.4/3.2 kPa) at the beginning of the procedure. While attempting to record a reliable pulmonary wedge pressure, the main pulmonary artery pressure was observed to vary spontaneously, finally dropping to 18/8 mmHg (2.4/1.1 kPa) about 30 minutes after the first measurement was obtained. At that time, the mean pulmonary wedge pressure was 9 mmHg (1.2 kPa), with a *v* wave of 14 mmHg (1.9 kPa). The systemic blood pressure as measured by sphygmomanometer was unchanged at 110/70 mmHg (14.6/9.3 kPa); the heart rate was 120/minute. The murmur was barely audible.

Phenylephrine, 1 mg diluted in 10 ml of normal saline, was injected intravenously. Within 1 minute, the blood pressure rose to 150/90 mmHg (19.9/11.9 kPa) and the heart rate dropped to 100/minute. A giant *v* wave appeared on the pulmonary wedge tracing (Fig. 1) while the pulmonary artery pressure rose to 84/30 mmHg (11.2/3.9 kPa) (Table). The patient complained of chest pain and of increasing dyspnoea. The murmur became much louder. These changes persisted for at least 30 minutes though the blood pressure had dropped to its previous level.

[1] Present address: Division of Cardiology, Stanford University School of Medicine, Stanford, California, U.S.A.

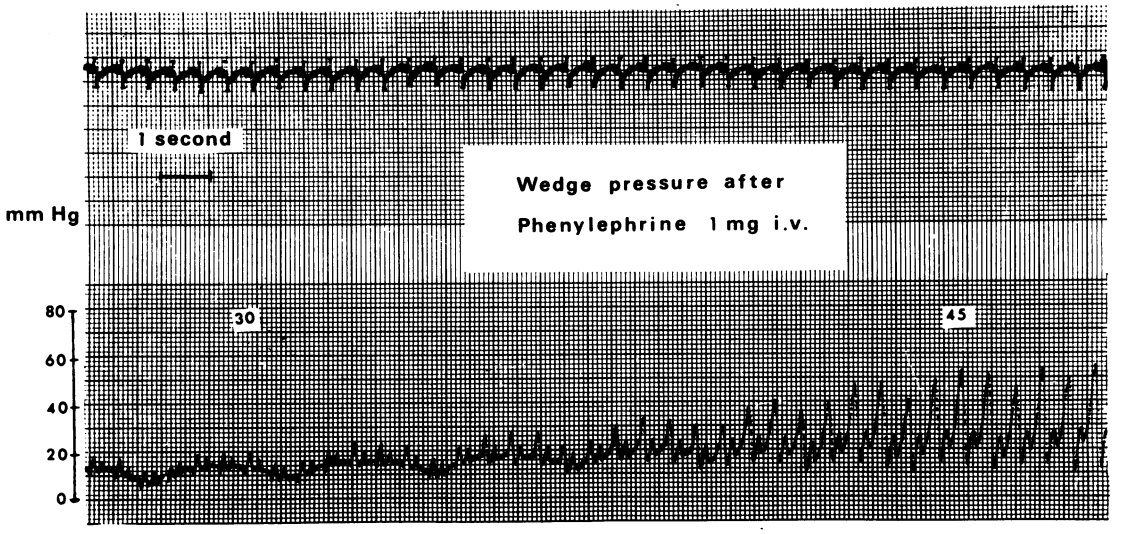


FIG. 1 *Continuous recording of the electrocardiogram* (above) *and of the pulmonary wedge pressure* (below) *after phenylephrine injection, 1 mg intravenously. Paper speed is 10 mm/s. 30 and 45 seconds after drug injection are marked. The* v *wave reached 64 mmHg (8.5 kPa) after 2 minutes.*

TABLE *Haemodynamic data before and after injection of phenylephrine, 1 mg intravenously*

| | *Before phenylephrine* | | *After phenylephrine* | | |
|---|---|---|---|---|---|
| | *0* | *30 min* | *2 min* | *30 min* | *60 min* |
| Right atrium (mmHg) | — | (4)<br>*(0.5)* | (10)<br>*(1.3)* | (12)<br>*(1.6)* | — |
| Right ventricle (mmHg) | — | 34/6<br>*4.5/0.8* | 80/8<br>*10.6/1.1* | 80/11<br>*10.6/1.5* | — |
| Pulmonary artery (mmHg) | 56/24<br>*7.4/3.2* | 18/8<br>*2.4/1.1* | 84/30<br>*11.2/3.9* | 84/30<br>*11.2/3.9* | — |
| Pulmonary wedge (mmHg) | — | (9)<br>*(1.2)* | (32)<br>*(4.2)* | (38)<br>*(5.0)* | — |
| *a* wave (mmHg) | — | 12<br>*1.6* | 23<br>*3.0* | 30<br>*3.9* | — |
| *v* wave (mmHg) | — | 14<br>*1.9* | 64<br>*8.5* | 60<br>*7.9* | — |
| Aorta (mmHg) | — | — | — | — | 90/55<br>*11.9/7.3* |
| Left ventricle (mmHg) | — | — | — | — | 90/20<br>*11.9/2.6* |
| Brachial cuff (mmHg) | 110/70<br>*14.6/9.3* | 110/70<br>*14.6/9.3* | 150/90<br>*19.9/11.9* | 110/70<br>*14.6/9.3* | — |

Figures in parentheses indicate mean pressures.
Pressures in italics are in kPa units.

The catheter was withdrawn and it was planned to stop the procedure. Within a few minutes however the chest pain disappeared. The murmur diminished in intensity and the patient became much less dyspnoeic. No changes were observed in the blood pressure or heart rate.

*Left heart catheterization* was then performed (Table), followed by a left ventricular cineangiography in the right anterior oblique projection which showed a mild to moderate degree of mitral regurgitation into an enlarged left atrium. The left ventricle was moderately enlarged and contracted only poorly.

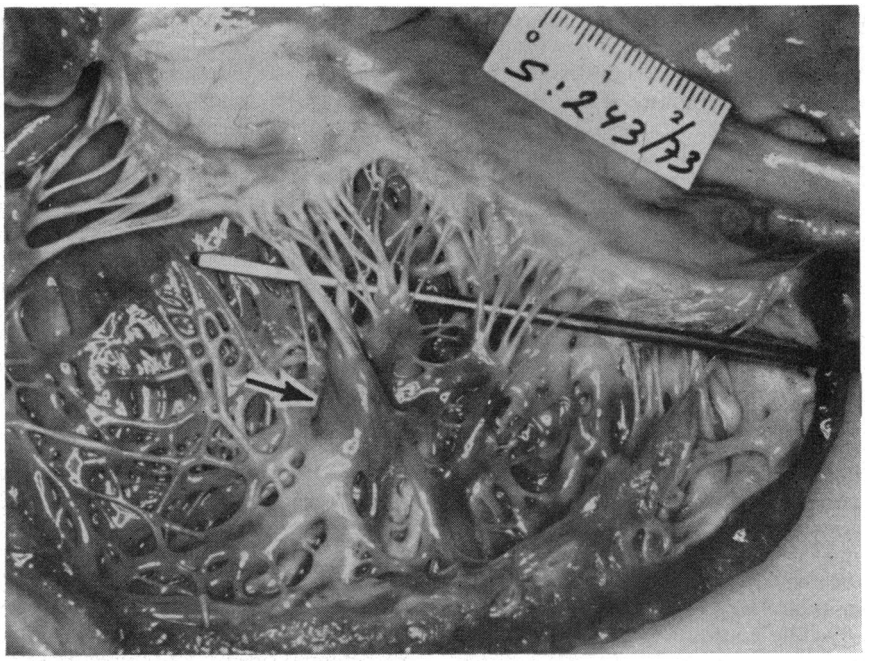

FIG. 2 *View of the left ventricle showing the mitral valve and the myocardial infarction involving the posterior papillary muscle* (arrow).

*Coronary arteriography* was attempted 2 weeks later. After the first injection in the left coronary artery, the patient developed complete heart block with shock and died despite all resuscitation efforts.

*Postmortem examination of the heart* showed diffuse, severe coronary atherosclerosis, an old anteroseptal infarction, and an old extensive posterolateral infarction involving the posterior papillary muscle (Fig. 2). The mitral valve leaflets and chordae appeared normal.

## Discussion

Before cardiac catheterization, our patient was thought to suffer from acute subvalvar mitral incompetence. The finding of a normal pulmonary wedge pressure would be unusual in this condition (Raftery, Oakley, and Goodwin, 1966) and was not expected in view of the clinical and radiological findings. However, the extent to which the pulmonary wedge *v* point increased after injection of phenylephrine was clearly disproportionate to the rise in systolic pressure and strongly suggested that severe mitral regurgitation was present under stress conditions (Braunwald, Welch, and Morrow, 1958). Of interest was the fact that the increase in the intensity of the systolic murmur and the giant *v* wave persisted long after the blood pressure and the heart rate had returned to their previous value, suggesting that various levels of mitral regurgitation occurred independently of changes in the afterload. The *a* wave on the pulmonary wedge tracing also increased after the injection of phenylephrine, though proportionally much less than the *v* wave. This probably reflected a rise in the filling pressure of the left ventricle. The spontaneous drop in the pulmonary artery pressure coincident with a reduction in the intensity of the murmur observed at the beginning of the study probably reflected varying degrees of severity of mitral regurgitation. Unfortunately no reliable wedge pressure could be obtained during this period. The character of the chest pain which occurred after the injection of phenylephrine was similar to that described during the previous episodes of acute pulmonary oedema. It probably represented myocardial ischaemia though acute distension of the left atrium could be implicated.

The necropsy revealed that an extensive lesion of the posterior papillary muscle caused by myocardial infarction was responsible for the mitral valve dysfunction.

It is often difficult to assess whether heart failure is caused mainly by myocardial damage or by the subvalvar lesion in this situation, but we believe

that these findings support the view that intermittent severe mitral regurgitation caused by papillary muscle dysfunction contributed significantly to the recurrent episodes of heart failure presented by our patient. Increased mitral regurgitation provoked either by systemic hypertension or presumably by an increased degree of papillary muscle dysfunction would precipitate severe heart failure. A lesser degree of papillary muscle dysfunction would be associated with normal pulmonary artery and pulmonary wedge pressures, and with a reduced intensity of the systolic murmur as observed during catheterization. The left ventricular cineangiogram was performed while the systolic murmur was reduced in intensity, and presumably less mitral regurgitation was present. While giving valuable information on the ventricular function it probably did underestimate the level of regurgitation occurring under stress conditions.

It is well known that mitral regurgitation caused by papillary muscle dysfunction may be present as a fixed lesion or may exist as a changing disorder (Burch, DePasquale, and Phillips, 1968). In some patients the systolic murmur can be heard only during an episode of chest pain. In one reported case, the pain and the murmur were followed by the development of acute pulmonary oedema believed to be secondary to a sudden free leakage of blood into a non-compliant left atrium (Holmes, Logan, and Winterbottom, 1968).

Few data are available to document that varying degrees of mitral regurgitation caused by papillary muscle dysfunction can cause significant changes in the haemodynamic and clinical status of a given patient (Brody and Criley, 1970; Lipp *et al.*, 1972), and in none of these reports was anatomical evidence of the papillary muscle lesion presented. One major implication of these studies is that some patients with intermittent acute mitral incompetence responsible for acute heart failure can show no or minimal evidence of regurgitation when studied in the laboratory. Unless haemodynamic changes suggesting acute mitral regurgitation develop spontaneously during catheterization, the dynamic and potentially serious character of the subvalvular mitral incompetence is likely to be underestimated.

No provocative test designed to increase specifically the degree of papillary muscle dysfunction is presently available. Braunwald *et al.* (1958) used pharmacological agents to increase acutely resistance to ventricular ejection. They suggested that the extent to which the left atrial *v* point was increased during norepinephrine infusion could be of diagnostic value in detecting mitral insufficiency. Agents producing hypertension without increasing myocardial contractility appear to provide a more sensitive test than norepinephrine for the presence of mitral regurgitation (Braunwald *et al.*, 1958; Ross, Cooper, and Lombardo, 1960). For a given regurgitant volume, the height of the *v* wave will depend on the left atrial size and on its pressure-volume relation (Ross *et al.*, 1960). Vasopressor drugs have been injected into a great variety of patients with mitral regurgitation, including patients with papillary muscle dysfunction (Heikkilä, 1967). Most showed a typical increase in the amplitude of the *v* wave on the pulmonary wedge tracing, and an enhancement of the murmur intensity. In none of these patients were spontaneous variations of the systolic murmur or in the heart pressures noted at the time of catheterization.

These data and the findings in our patient suggest that vasopressor drugs may be of use when the syndrome of acute intermittent mitral regurgitation is suspected but not demonstrated during routine cardiac catheterization. While not reproducing the haemodynamic and clinical changes occurring spontaneously, vasopressor drugs may 'unmask' the potential severity of the regurgitation and could help in selecting those patients in need of surgical therapy.

## References

Braunwald, E., Welch, G. H., and Morrow, A. G. (1958). The effect of acutely increased systemic resistance on the left atrial pressure pulse: a method for the clinical detection of mitral insufficiency. *Journal of Clinical Investigation*, **37**, 35.

Brody, W., and Criley, J. M. (1970). Intermittent severe mitral regurgitation. Hemodynamic studies in a patient with recurrent acute left-sided heart failure. *New England Journal of Medicine*, **283**, 673.

Burch, G. E., DePasquale, N. P., and Phillips, J. H. (1968). The syndrome of papillary muscle dysfunction. *American Heart Journal*, **75**, 399.

Heikkilä, J. (1967). Mitral incompetence as a complication of acute myocardial infarction. *Acta Medica Scandinavica*, Suppl. 475, 7.

Holmes, A. M., Logan, W. F. W. E., and Winterbottom, T. (1968). Transient systolic murmurs in angina pectoris. *American Heart Journal*, **76**, 680.

Lipp, H., Gambetta, M., Schwartz, J., de la Fuente, D., and Resnekov, L. (1972). Intermittent pansystolic murmur and presumed mitral regurgitation after acute myocardial infarction. *American Journal of Cardiology*, **30**, 690.

Raftery, E. B., Oakley, C. M., and Goodwin, J. F. (1966). Acute subvalvar mitral incompetence. *Lancet*, **2**, 360.

Ross, J., Cooper, T., and Lombardo, C. R. (1960). Hemodynamic observations in experimental mitral regurgitation. *Surgery*, **47**, 795.

Requests for reprints to Dr. E. Riss, Department of Cardiology, Rambam University Hospital, Haifa, Israel.